Type A behaviour pattern: a concept revisited

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It is generally accepted that the type A behaviour pattern is a risk factor in the development of coronary artery disease (CAD). Type A people have been characterized as hard-driving, competitive, aggressive and hurried. A number of investigators have attempted to correlate these facets of type A behaviour with increased risks of CAD. However, there have been conflicting results, primarily owing to differences in methods and CAD outcomes and inconsistencies associated with measuring the type A behaviour pattern. As a result, researchers have begun to focus on subcomponents of the type A behaviour pattern, particularly hostility and anger, that appear to be more reliable predictors of CAD outcome. A reconceptualization of the type A behaviour pattern is required.

On croit généralement que le risque de coronaropathie est particulièrement grand chez les personnes qui ont un comportement dit du type A: pressé, agressif, en tension constante vers le succès et poussé par la concurrence. Mais les divers chercheurs qui ont voulu mettre ces traits en rapport avec le risque de coronaropathie ont obtenu des résultats contradictoires, à cause surtout de différences méthodologiques, de la variabilité du pronostic des coronaropathies et de divergences dans l'appréciation des composantes du susdit type A. On en est venu à considérer surtout certains de ses aspects, telles l'hostilité et la colère rentrée, qui semblent reliés de plus près à ce pronostic. Il faut donc adopter un nouveau concept du comportement de type A.

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 \P he type A behaviour pattern, as defined by Friedman and Rosenman,1 is generally regarded as an important risk factor in coronary artery disease (CAD). Traditional risk factors such as smoking, obesity, hypertension and diabetes mellitus account for no more than 50% of variance with respect to CAD outcomes such as angina and myocardial infarction. Both prospective and retrospective epidemiologic studies have shown a strong association between the type A behaviour pattern and myocardial infarction.2-5 As well, the conclusions of a review panel convened by the US National Heart, Lung, and Blood Institute of the National Institutes of Health confirmed the link between the type A behaviour pattern and CAD.6

Type A people are characterized as engaging in a chronic struggle to obtain an unlimited number of things from the environment in the shortest time or against opposing efforts. They exhibit a cluster of behaviours: increased competitiveness, a striving for achievement, aggressiveness that may be strongly repressed, impatience, restlessness, hyperalertness, explosive speech stylistics and a chronic sense of urgency. These behaviours do not stem solely from personality factors but, rather, develop from an interaction between certain personality attributes and environmental challenges. People who display these behaviours appear to have a predisposition to respond to certain stressors in a particular pattern. According to Rosenman and colleagues² there are various degrees of intensity of type A behaviour, ranging from type A1 (fully developed A) to A2 (weak A). Those who do not exhibit these behaviours are labelled type B and do not show increased risk for CAD. They are typically more relaxed, easygoing, satisfied and unhurried. Similarly, they may be categorized as type B3 (weak B) to B4 (fully developed B). People who do not fall into either category are classified as type X. However, most practitioners use a simple dichotomous A/B classification.

Because type A people are generally regarded as highly productive and achievement oriented, type B people are often seen as nonproductive and lax about work output. This is an unfortunate misconception. Unlike type A people, type B people do not possess the drive to control and master their environment. However, their level of motivation does not differ, nor does their work output. They simply approach tasks with a better-developed sense of inner security and adequacy. This enables them to be equally productive without the degree of competitiveness associated with fear of failure.

Since the end of the 19th century there have been a number of observations relating CAD to personality and behaviour patterns. Osler^{7,8} strongly associated stress and hard-driving behaviour with CAD. Similar psychobehavioural characteristics were subsequently noted by physicians such as Menninger and Menninger⁹ and Dunbar, ¹⁰ who also found this group of patients to have strongly aggressive tendencies and repressed hostility. Additionally, such patients were noted to be highly achievement oriented and to assume excessive amounts of responsibility.

The early psychosomaticists such as Dunbar argued psychogenic causality in CAD. Their work, however, suffered from a lack of objective predictor variables and prospective validation and from measurement weaknesses. Current research on the relation of psychologic factors and CAD differs markedly in terms of major refinements in measurement and research methods and in a theoretical framework that suggests that many factors are involved in the development of CAD. As well, current research is attempting to determine pathophysiologic mechanisms responsible for CAD.

Mechanism of disease

A number of theories have been presented to explain the physiologic mechanism by which the type A behaviour pattern results in CAD. One theory is that emotional stress results in β adrenergic stimulation, which leads to an increase in heart rate and cardiac output. This in turn causes elevated peripheral vascular resistance because of vasoconstriction.11 This increase in blood pressure may, over time, cause damage to the arterial wall through increased shearing forces, allowing plaque buildup to begin at the site of damage.12 It has been suggested that damage to the arterial wall causes the release of vasoconstrictive agents, which may, in periods of increased stress, result in arterial vasospasm, causing angina or even myocardial infarction.12

Another theory is that elevated catecholamine levels cause arterial damage, producing sites for platelet aggregation and adhesion.^{13,14} The caps of atheromatous plaque that are subsequently formed lead to arterial stenosis. Researchers have shown that plasma catecholamine levels increase in response to various stressors, ranging from public speaking¹⁵ to experimentally induced mental stress.^{16,17} What is still unknown is the degree to which psychologic stress activates these mecha-

nisms. Certainly, both retrospective evidence linking emotional stress to sudden death^{18,19} and prospective studies^{2,20} strongly support the existence of such a link.

Type A behaviour as a risk factor

Friedman and Rosenman¹ initially relied on observations of their patients' behaviour to develop the type A concept. They went on to create a structured interview protocol to measure type A behaviour. Subsequently they provided the first prospective evidence of an association of the type A behaviour pattern with CAD. In the Western Collaborative Group Study 3500 middle- and upper-level executives who were all initially free of CAD were classified as either type A or type B on the basis of the results of the structured interview. After 8.5 years of follow-up the men who were classified as type A had twice the rate of CAD as those classified as type B, even when other CAD risk factors were controlled for.² Type A behaviour was found to be associated with a risk of angina and myocardial infarction.

In the Framingham Study white-collar and blue-collar workers were investigated over an 8-year period.³ The type A behaviour pattern was found to be an independent predictor of angina and myocardial infarction in men aged 45 to 64 years and of CAD and angina in women of the same age. The French-Belgian Collaborative Group,²¹ using the Bortner Rating Scale,²² also showed that the type A behaviour pattern was a significant predictor of CAD, myocardial infarction and sudden death in 2800 men studied over 5 years. In addition, some angiographic studies have shown a correlation between the type A behaviour pattern and CAD;²³⁻²⁶ disease severity was determined by the degree of coronary atherosclerosis.

In spite of these positive findings, other recent prospective studies have failed to show an association between the type A behaviour pattern and CAD. In the Multiple Risk Factor Intervention Trial (MRFIT) both the structured interview of Rosenman and colleagues² and the Jenkins Activity Survey²⁷ were used to test the effect of a multifactor intervention program on death from CAD in men aged 35 to 57 years.²⁸ No association was found between type A behaviour and illness or death due to CAD.

The Aspirin Myocardial Infarction Study was designed to test the effect of regular administration of acetylsalicylic acid on death rates among people who had already suffered at least one myocardial infarction.²⁹ The Jenkins Activity Survey was used to assess the behaviour pattern. The scores were not predictive of recurrence of myocardial infarction or of death from CAD. In the Finnish twincohort study a shortened version of the Bortner Rating Scale was administered to 23 000 CAD-free men who were followed for 6 years.³⁰ The type A behaviour pattern was not found to be correlated

with death from all natural causes or from CAD alone. As well, angiographic studies did not show evidence of an association between the severity of coronary artery occlusion and the type A behaviour pattern.³¹⁻³⁵

Why is there such a discrepancy in results between the two groups of studies? One possible explanation is the inconsistencies associated with the measurement of type A behaviour. To date, four main instruments have been used: the structured interview, the Jenkins Activity Survey, the Framingham Type A Scale and the Bortner Rating Scale. All have been criticized as having a variety of flaws that limit their predictive power. While the conceptual definition of the type A behaviour pattern focuses on elements of speed and impatience, job involvement and hard-driving, competitive behaviour, the operational definition emphasizes speech stylistics such as loudness, explosivity, and rapidity of response, as measured by the structured interview. Even here, problems have been encountered. The proportions of MRFIT subjects classified as type A varied a great deal among the interviewers. While Rosenman was the ultimate judge of type A behaviour in the Western Collaborative Group Study, he assessed only 15% of the MRFIT subjects; this contributed to the lower rates of identification of the type A behaviour pattern and to the negative findings of the MRFIT study.

The structured interview has been used in almost half of the studies performed thus far; the Jenkins Activity Survey or the Bortner Rating Scale has been used in the other half.³⁶ As a result, it has been extremely difficult to assess the comparability of the measures and their power to predict cardiovascular outcome.

A number of problems impede the precise assessment of type A behaviour. First, the currently used measures are psychometrically imprecise, in that it is unclear which aspects of type A behaviour account for the classification of a person as type A. Type A behaviour is a complex set of behaviours, but assessment relies on self-reporting or observation of the general class of behaviours. Second, the existing measures do not account for the fact that different aspects of type A behaviour are related to different CAD outcomes. It is theorized that one or more pathophysiologic mechanisms are responsible for the various forms that CAD takes; therefore, relying on one measure alone may result in erroneous conclusions. Third, available measures of type A behaviour are context dependent and do not account for the specificity or frequency of interaction between the person and environmental challenges. This interaction must be considered in any assessment of type A behaviour.

Because of these difficulties, some researchers have begun to look at subcomponents of the type A behaviour pattern that appear to be more reliable in predicting CAD outcome. Two subcomponents that have been singled out as having predictive power are hostility and anger.

Hostility and anger

Hostility has been defined as a long-standing attitude of ill will and negative evaluations of people and events.³⁷ Anger has been defined as an emotional state consisting of feelings that vary in intensity from mild irritation or annoyance to rage and fury.38 The suggestion that anger and hostility are related to CAD had its origin in the work of Gaub, an 18th-century Dutch physician who noted excessive interpersonal anger in his patients with CAD.³⁹ In 1939 Alexander⁴⁰ developed a conceptual framework that was grounded in psychoanalytic theory. He posited that people with hypertension are caught between chronic passiveness and hostile impulses, and that repression of these impulses results in chronic tension. Although Alexander's work lacked methodologic rigour, his idea sparked a great deal of interest in the relation between personality factors and CAD. More recently, Spielberger and coworkers⁴¹ commented on the negative effect of anger and hostility on physical and mental health.

A number of assessment devices, ranging from interviews and projective tests to self-reported inventories and behavioural tests, have been used to measure anger and hostility.⁴² In general, measures of anger and hostility have acceptable psychometric properties, with reliability coefficients ranging from 0.50 to 0.80.

Recent research has resulted in the development of a protocol for testing mental stress.43 With this protocol people can be classified as "hot reactors" or "cold reactors", depending on their physiologic responses to stressful tasks such as mental arithmetic problems and computerized games. Such tasks are thought to elicit angry reactions in people with high scores on the "potential for hostility" index of the structured interview. It is not yet known whether hot reactors are at greater risk for CAD. No large-scale prospective studies have shown a correlation between cardiovascular reactivity and disease outcomes, although a number of studies have shown a relation between reactivity and hostility by means of various measures.26,44-46 This type of mental-stress testing may produce important information that will help clarify the mechanism by which type A behaviour affects disease development. Assessment of the relative contribution of overresponsivity may lead to a better understanding of the influence of anger and hostility on the development of disease.

Other prospective data are available on the relation of hostility and anger to CAD. Barefoot and associates⁴⁷ found that high levels of hostility were predictive of CAD and death from all causes in a 25-year study of 255 physicians who were initially assessed while in medical school. Hostility was measured with the Cook-Medley Hostility Inventory,⁴⁸ a subscale derived from the Minnesota Multiphasic Personality Inventory.⁴⁹ In the Western Electric Study, Cook-Medley Hostility Inventory scores were found to be related to the 10-year

incidence of major CAD events such as myocardial infarction and death from CAD.⁵⁰ Additionally, Williams and collaborators²⁶ found that patients with Cook-Medley Hostility Inventory scores above the sample median were more likely than those with scores below the sample median to have 75% or greater stenosis of at least one coronary artery, as measured by coronary angiography.

The findings of prospective studies in which other measures of anger were used are consistent with these findings. In the Framingham Study multivariate analysis of the 8-year incidence of CAD showed that scales measuring "not showing anger outwardly" and "not discussing anger" were predictive of CAD in both men and women.3 In addition, analysis of subcomponents of the type A behaviour pattern, as measured with the structured interview, has shown that certain items tap the global index "potential for hostility", which is based on harsh responses, the use of obscenities and rudeness. In one study "potential for hostility" scores distinguished men with silent myocardial infarction from controls matched for age and occupation.51 More recently, Dembroski and colleagues³¹ attempted to determine, through subcomponent scores on the structured interview, which elements of the type A behaviour pattern were related to CAD severity. Their findings indicated that only "potential for hostility" and "anger-in" (internalized anger) were significantly and positively associated with disease severity. Similar findings were recently reported by MacDougall and collaborators.52 In contrast, McCranie and coworkers⁵³ found that higher hostility scores were not predictive of CAD or of death from all causes. These authors commented that various components of hostility must be carefully assessed to determine which are most strongly and consistently associated with CAD.

While these findings suggest that anger and hostility may be the essential components of the type A behaviour pattern that predispose to CAD, it is unclear what the measures used to assess anger and hostility are specifically tapping. It is well known that anger and hostility are composed of many elements that likely affect the development of CAD. However, it is unclear which behavioural mechanisms are responsible for the increased risk of CAD. The increased risk may be due to anger, which is one of a number of indicators of emotionality. This hypothesis is consistent with data linking anxiety and depression to poor cardiovascular health.54 Another possibility is that anger and hostility may reflect a more general style of social incompetence. People may be at increased risk if they want to be with others but lack the social skills to do so successfully, which is apt to result in frustration and anger. Further research is necessary to validate the measures currently in use so that the role of anger and hostility in the development of CAD may be more clearly delineated.

Intervention strategies

As the concept of the type A behaviour pattern gained acceptance over the years, numerous intervention strategies were developed to treat type A people. Stress management approaches involving behaviour modification strategies have had generally positive short-term outcomes.⁵⁵⁻⁵⁹ However, the variety of research designs and methods has made comparisons of treatment efficacy difficult. As well, problems in operationally defining the type A behaviour pattern have hindered the development of specific treatment programs. For instance, it is not practical to treat all aspects of the type A behaviour pattern, nor is it necessary, as certain people are not at increased risk for CAD in spite of being labelled type A.

When the type A behaviour pattern is defined according to the original features, it shows poor specificity as a predictor of CAD. Only a small proportion of those identified as type A exhibit symptoms of CAD. Current research suggests that this poor specificity may be due to the fact that only certain components of the behaviour pattern are relevant. Consequently, in designing treatment programs one must consider the components that are predictive of an increased risk of CAD. Since anger and hostility have been identified as key factors, treatment approaches that focus on the management of these states have been developed. 60,61

Anger management is a cognitive behaviour therapy approach based on well-known stress inoculation techniques.⁶² It is founded on the belief that the intensity of anger and hostility reactions is determined on a cognitive level by the appraisals, attributions and expectations of a particular situation. Tension and fatigue further influence the anger response, resulting in withdrawal or antagonism. Thus, anger is a function of environmental cues and the appraisal of one's overt and covert behaviour. Treatment consists of teaching patients cognitive, affective and behavioural coping skills and subsequently exposing them to regulated levels of stress so that they can practise their newly acquired skills.

Conclusion

The predictive power of the global type A concept appears to be fading as evidence accumulates in support of subcomponent analysis of the type A behaviour pattern. Anger and hostility must now be seen as the critical aspects that predispose to CAD. A reconceptualization is thus required so that these components are given more weight. Effective and specific treatments need to be further tailored to reduce behaviours that may carry an increased risk for the development of CAD.

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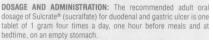
These interactions appear to be non-systemic and to result from the binding of Sulcrate® to the concomittantly administered drug in the gastro-intestinal tract. In all cases, complete bioavailability was restored by separating the administration of Sulcrate® from that of the other agent by 2 hours

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